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# Event-rate effects in the flanker task: ERPs and task performance in children with and without AD/HD

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## Publication Details

Johnstone, S. J. & Galletta, D. (2013). Event-rate effects in the flanker task: ERPs and task performance in children with and without AD/HD. *International Journal of Psychophysiology*, 87 (3), 340-348.

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# Event-rate effects in the flanker task: ERPs and task performance in children with and without AD/HD

## Abstract

Demanding tasks require a greater amount of effort, in which case individuals are required to alter their energetic-state to a level appropriate to perform the task. According to the Cognitive-Energetic Model (CEM), children with AD/HD are unable to effectively modulate their energetic state, leading to task underperformance. Using an Eriksen flanker task with varying event-rates, the current study compared the ability of typically-developing children and children with AD/HD to modulate their energetic state. In line with the CEM, it was predicted that children with AD/HD would underperform in the fast and slow event-rates. Results indicated that the groups did not differ in commission errors (i.e., incorrect responses). However, children with AD/HD made more omission errors to incongruent stimuli at the fast and slow event-rates, compared to controls. N2 amplitude was significantly larger for the AD/HD than control group in the slow event-rate. It is concluded that the energetic state modulation dysfunction in children with AD/HD results in an inability to attend to the task, as opposed to an inability to perform the task itself. Furthermore, these task performance differences did not manifest in either the N2 or P3 ERP components. Therefore, inattention in children with AD/HD may have its locus in response preparation, as opposed to stimulus processing, but more research is required to validate these conjectures.

## Keywords

children, without, ad, performance, hd, erps, task, flanker, effects, rate, event

## Disciplines

Education | Social and Behavioral Sciences

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Manuscript Number: CCD1238

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Article Type: Special Issue: Cognitive Control

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## Highlights

- Children with AD/HD unable to effectively modulate energetic state
- Event-rate varied during interference control in children with and without AD/HD
- Omission but not commission errors more frequent in children with AD/HD
- N2 larger for AD/HD than control group in slow event-rate
- Apparent inability to attend to task, as opposed to perform the task

**Title:** Event-rate effects in the flanker task: ERPs and task performance in children with and without AD/HD

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## **Abstract**

Demanding tasks require a greater amount of effort, in which case individuals are required to alter their energetic-state to a level appropriate to perform the task. According to the Cognitive-Energetic Model (CEM), children with AD/HD are unable to effectively modulate their energetic state, leading to task underperformance. Using an Eriksen flanker task with varying event-rates, the current study compared the ability of typically-developing children and children with AD/HD to modulate their energetic state. In line with the CEM, it was predicted that children with AD/HD would underperform in the fast and slow event-rates. Results indicated that the groups did not differ in commission errors (i.e., incorrect responses). However, children with AD/HD made more omission errors (i.e., impulsively early or non-responses) to incongruent stimuli at the fast and slow event-rates, compared to controls. N2 amplitude was significantly larger for the AD/HD than control group in the slow event-rate. It is concluded that the energetic state modulation dysfunction in children with AD/HD results in an inability to attend to the task, as opposed to an inability to perform the task itself. Furthermore, these task performance differences did not manifest in either the N2 or P3 ERP components. Therefore, inattention in children with AD/HD may have its locus in response preparation, as opposed to stimulus processing, but more research is required to validate these conjectures.

## **Keywords**

Children, AD/HD, ERPs, flanker, interference, event-rate

## 1. Introduction

AD/HD is a developmental neuropsychopathology characterised by age-inappropriate levels of inattention (the Predominantly Inattentive Type), hyperactivity and impulsiveness (the Predominantly Hyperactive-Impulsive Type), or a combination of both types (the Combined Type; American Psychiatric Association, 2000). AD/HD is one of the most widespread childhood psychiatric conditions, with epidemiological studies estimating a prevalence of between 5% and 10% (Faraone et al., 2003; Polanczyk et al., 2007; Skounti et al., 2007).

Until recently, neuropsychological models of AD/HD converged upon the notion that an inhibitory deficit constitutes the pathogenesis of the disorder (e.g. Barkley, 1997; Quay, 1997). Whilst there is no doubt that inhibition is impaired in those with AD/HD (see Nigg, 2001), these ‘fixed-deficit’ models fail to account for the heterogeneous symptomatology of the disorder (Sergeant et al., 2002). Thus, models have emerged that account for the heterogeneity of AD/HD by taking into account both computational and state factors. One such model is the Cognitive Energetic Model (CEM; Sergeant, 2000). Based on early work by Sanders (1983), the CEM theorises that energetic factors such as arousal, activation and effort mediate task performance. Tasks that are highly demanding require a great amount of energy to perform successfully, in which case the individual is not likely to be in the appropriate energetic state. The individual must then increase effort to mobilise energetic resources in order to equilibrate their energetic state with the energetic requirements of the task (Kok, 2001; Robert and Hockey, 1997). According to the CEM, in situations of sub-optimal energetic state, individuals with AD/HD cannot successfully adjust their energetic state to the level required by the task.

Research has revealed that this dysfunction originates in the processing stage involved in generating motor responses (Sergeant, 2005; Sergeant and Scholten, 1985). Event-rate (i.e., the speed at which stimuli are presented) varies the rate at which motor responses are required, making it an appropriate manipulation of energetic state (Sanders, 1983). Compared to a medium event-rate, a fast event-rate is thought to induce over-arousal and fast inaccurate responding, and a slow event-rate is thought to induce under-arousal and slow inaccurate responding (Sergeant, 2000; Sonuga-Barke et al., 2010). Thus, the CEM predicts that the effect of arousal-level on task performance should follow a U-shaped pattern.

Inhibition is a multifaceted process, with behavioural inhibition consisting of suppressing the initial proponent response to an event, interrupting an activated response, and interference control (Barkley, 1997). The flanker task is thought to measure interference control (Sanders and Lamers, 2002), requiring the participant to attend to task-relevant stimuli whilst concurrently inhibiting task-irrelevant

stimuli (van Veen et al., 2001). Incongruent flankers provide response information that conflicts with the response information of the target stimuli (i.e., one requires a left-handed response and the other requires a right-handed response), resulting in an interference effect that is characterised by a slower reaction time (RT) and a greater number of errors (Kopp et al., 1996). Conversely, in the congruent condition the target stimulus and the flanking stimuli provide complementary response information (i.e., they both denote the same response) that facilitates a reduction in RT and errors (Flowers, 1990).

In the flanker task, children with AD/HD show slower RTs and lower response accuracy than typically-developing children for all stimulus types (Johnstone et al., 2010; Jonkman et al., 2007; Konrad et al., 2006). Compared to typically-developing children, children with AD/HD also show a more delayed RT to incongruent stimuli compared to neutral stimuli (Jonkman et al., 2007; Ridderinkhof et al., 2005).

The present study aimed to examine the ability of children with AD/HD to modulate their energetic state by manipulating the event-rate of the Eriksen flanker task (Eriksen and Eriksen, 1974). In addition to task performance indices such as reaction time (RT) and error-rates, the present study will also employ event-related potentials (ERPs), with a focus on the N2 and P3 ERP components. The N2, or “control N2”, is a fronto/fronto-central negativity occurring between 200 and 400 ms after stimulus presentation in children (Folstein and Van Petten, 2008; Johnstone et al., 2010). In the flanker task the N2 is enhanced to incongruent stimuli compared to other stimuli (Heil et al., 2000; Johnstone et al., 2009; Johnstone et al., 2010; Kopp et al., 1996; Yeung et al., 2004). Furthermore, the N2 is insensitive to stimulus expectancy (Gehring et al., 1992) and stimulus probability (Bartholow et al., 2005). Together these results suggest that the N2 is an index of the processing of conflicting response information, a theory that is supported by evidence of the N2 in other conflict-related paradigms such as the Stroop task (Liotti et al., 2000; West and Alain, 2000) and stop-signal task (Band et al., 2003).

The P3 is a centro-parietal positivity occurring between 300 and 600 ms after stimulus presentation in children (Broyd et al., 2007; Johnstone et al., 2008; Polich, 2007). A parietally maximal P3 component has been identified in a variety of paradigms whose latency has been associated with the time taken to evaluate a stimulus (Polich and Kok, 1995) and amplitude related to the amount of information transferred to working memory (Kok, 2001; Polich and Kok, 1995; Wiersema et al., 2006a). In the flanker task this component is maximal centro-parietally, and is enhanced and delayed to incongruent stimuli compared to other stimulus types in both adults (Folstein and Van Petten, 2008; Ridderinkhof and van der Molen, 1995) and children (Johnstone et al., 2009; Rueda et al., 2004).

There is only one study to date that directly manipulates event-rate in the flanker task. van Meel, Heslenfeld, Oosterlaan and Sergeant (2007) employed a flanker task with both a fast and a



slow event-rate to examine children's ability to control their response style (i.e., the speed/accuracy trade-off). Results showed a faster RT and a larger number of errors in the fast compared to the slow event-rate. However, the study did not include a moderate event-rate to test the CEM's predicted U-shaped pattern of task performance. Furthermore, the study lacked appropriate measures of arousal to confirm the successful manipulation of energetic state and examined neither the N2 nor the P3 ERP components.

Event-rate manipulations in other inhibition-related tasks may also be informative for the present study. van der Meere, Gunning and Stermerdink (1999) varied the event-rate in the Go/Nogo task in a clinical trial of pharmacological treatments for children with AD/HD. Consistent with the U-shaped pattern of performance predicted by the CEM, results showed that total errors were larger in the fast and slow event-rate than in the medium event-rate. Additionally, Benikos and Johnstone (2009) included a control group in their manipulation of Go/Nogo event-rate. They found that whilst typically-developing children showed a fast>medium>slow trend for Nogo errors, children with AD/HD made more Nogo errors in the fast and slow event-rate than in the medium event-rate. Moreover, they found that the event-rate manipulation didn't affect the amplitude or latency of the N2 component, but the P3 component was larger at the fast event-rate than the slow event-rate for the control group but didn't differ between event-rates for the AD/HD group. Although the results of both studies are consistent with predictions made by the CEM, they examined a different type of inhibition using a different paradigm and neither included measures of arousal to confirm the successful manipulation of energetic state.

Another major limitation of previous research is that no measures of energetic state were used to confirm that the event-rate manipulation was successful. One method of measuring energetic state is through the analysis of rhythmic oscillations in the ongoing EEG. Research focused on oscillations in the alpha band (8-13Hz) has revealed an inverse relationship between alpha power and cortical arousal (Oakes et al., 2004; Shagass, 1972). More recently it has been found that alpha in the prestimulus period varies as a function of the energetic requirements of a task (Carp and Compton, 2009; Klimesch et al., 1998). As such, phasic changes in alpha power are taken as a functional negative correlate of arousal, a position supported by studies using electrophysiological and electrodermal measures of energetics (e.g. Barry et al., 2007; Barry et al., 2004). Therefore, the present study will also include a measure of pre-stimulus alpha power in order to confirm that successful manipulation of energetic state.

In sum, it was expected that, compared to the control group, the AD/HD group would perform worse at the fast and slow event-rates. It was also predicted that children with AD/HD and typically-developing children would show ERP component differences. The N2 component is

thought to index the level of conflict-related processing, therefore, children with AD/HD were expected to show a smaller incongruent > neutral N2 amplitude effect than typically-developing children. Furthermore, because the present study manipulates motor generation requirements as opposed to inhibitory requirements, this incongruent > neutral N2 effect was not expected to differ between event-rates. It was predicted that children with AD/HD would show a more enhanced and delayed P3 to incongruent stimuli than typically-developing children. Furthermore, the shorter time allowed for stimulus evaluation in the fast event-rate was predicted to reduce the latency and increase the amplitude of the P3 component across conditions, and this effect was predicted to be smaller in the AD/HD group. It was predicted that pre-stimulus alpha power should be larger for the fast event-rate and smaller for the slow event-rate, compared to the medium event-rate. Additionally, the CEM theorises that children with AD/HD cannot effectively regulate their energetic state. It was predicted that children with AD/HD will be over aroused at the fast event-rate and under aroused at the slow event-rate compared to typically-developing children.

## **2. Materials and Method**

### **2.1 Participants**

Twenty-nine children participated in this study. The AD/HD group consisted of nine males and five females, aged between 7 years 10 months and 14 years 7 months ( $M = 11.09$ ,  $SD = 2.15$ ). All children in the AD/HD group were diagnosed with AD/HD of the Combined Type by a psychologist according to the Diagnostic and Statistical Manual of Mental Disorders Version IV Text Revision (DSM-IV-TR; American Psychiatric Association, 2000) diagnostic criteria. Of the 14 children in the AD/HD group, 12 were taking treatment medication for AD/HD (10 methylphenidate, two dextroamphetamine) but all abstained from medication 24 hours (at least five half-lives for methylphenidate and two half-lives for dextroamphetamine; Pelham et al., 1990) prior to testing.

The control group consisted of 10 males and five females, aged between 7 years 6 months and 14 years 11 months ( $M = 11.66$ ,  $SD = 2.11$ ). Children in the control group were excluded if they scored in the borderline or clinical range (above 60) on the Conner's Parent Rating Scale – Revised. Furthermore, children in either group were excluded if they: (a) had an estimated IQ of less than 80, (b) had disorders of consciousness or head injuries, or (c) had any comorbid mental disorders. All children had normal or corrected-to-normal vision.

Irrespective of performance each child was given a certificate and a small chocolate bar for participating in the study. Parents received \$30 to compensate for travelling expenses and other costs incurred, and were given a written report on their child's spelling, reading and cognitive ability.

## **2.2 Stimuli**

Stimuli in the flanker task consisted of standard ASCII characters. Participants were required to respond to a central target stimulus that was either a left (<) or right (>) facing arrow. Target stimuli were either presented alone, or surrounded by flankers that were congruent (e.g. >>>>>), incongruent (e.g. <<><<) or neutral (e.g. ==>==) to the target stimuli. Each stimulus was presented with equal probability and in a pseudorandom order, with no two stimuli of the same type presented in succession. Stimuli subtended 2.3° of visual angle.

The flanker task was explained verbally and the participant was given the opportunity to read the on-screen instructions. The participant began each experimental task when ready. Each trial began with a central fixation cross (+) which remained on-screen for 500 ms. The fixation cross was immediately followed by a stimulus which remained on-screen until the participant made a response or for 1500 ms if no response occurred. Following the stimulus was a blank screen that lasted between 250 - 750 ms ( $M = 500$  ms) in the fast condition, 2750 - 3250 ms ( $M = 3000$  ms) in the medium condition, or 5750 - 6250 ms ( $M = 6000$  ms) in the slow condition.

There was a 16 trial practice block followed by three experimental blocks (fast, medium, slow) of 120 trials. The three experimental blocks were counterbalanced, such that each participant was randomly assigned a different order of experimental blocks. Participants were given a short break between blocks.

Participants were instructed to focus on the target stimulus and respond with a left or right handed button press according to the direction of the target arrow. Left-handed responses were made with the left “alt” button and right handed responses were made with the right “ctrl” button on a standard QWERTY keyboard. Responses that occurred between 200 ms and 1500 ms were considered valid and only the first response was recorded, otherwise they were considered errors of omission.

## **2.3 Electrophysiological Recording**

Continuous EEG data were recorded using Ag/AgCl electrodes from 19 scalp sites (FP1, FP2, Fz, F3, F4, F7, F8, Cz, C3, C4, C7, C8, Pz, P3, P4, T3, T4, T5, T6, O1, O2) placed according to the International 10/20 System. Participants were grounded by a cap electrode situated midway between Fz and FPz and referenced to digitally-linked ears. Only the midline electrode sites (Fz, Cz, Pz) were examined in the present study. Electro-oculogram (EOG) data were recorded using tin cup electrodes placed on the outer lateral canthi of the left (E3) and right (E4) eyes for horizontal eye movements, and 1cm above (E1) and below (E2) the left eye for vertical eye movements. Vertical EOG (vEOG) was quantified as E1-E2.

Impedance levels were below 5 k $\Omega$  for scalp electrodes and below 3 k $\Omega$  for reference and EOG electrodes at the beginning of testing. Data were amplified 19 times, sampled at 500 Hz, and digitally bandpass filtered (3 dB down, 24 dB/octave roll-off) at 0.1 and 100 Hz, using a NuAmps 40 channels amplifier system from Neuroscan (Compumedics Limited, Melbourne, Australia). Continuous EEG and EOG data were recorded for offline analysis.

## ***2.4 Data Quantification***

Data were analysed offline using the Neuroscan (Scan 4.3.1) software package. Time domain data were divided into epochs defined as 100 ms pre-stimulus to 900 ms post-stimulus onset and limited to correct responses only. Epochs were baseline corrected using the average amplitude in the 100 ms pre-stimulus period. An ocular artefact rejection procedure based on the vEOG channel (Semlitsch et al., 1986) was used to reduce the effect of eye movements and an artefact rejection procedure removed trials with activity exceeding  $\pm 120$   $\mu$ V in the F3, F4 or Fz sites.

The quantification of N2 and P3 data were treated differently. In line with Yeung and colleagues (e.g. Yeung et al., 2004; Yeung and Nieuwenhuis, 2009), quantification of the N2 component included bandpass filtering (zero phase shift, 2-12 Hz, 48 dB/oct roll-off) the data in order to eliminate the effect of slow parietal positive or negative shifts that may obscure the effects of interest. Alternatively, P3 data were low pass filtered (zero phase shift, 0.5-30 Hz, 48 dB/oct roll-off) in accordance with previous studies from our lab (e.g. Benikos and Johnstone, 2009; Johnstone et al., 2008; Johnstone et al., 2009; Johnstone et al., 2010).

Grand average ERP waveforms for each stimulus type and event-rate were examined to determine appropriate component latency ranges. ERP component peaks were automatically quantified using an automatic peak-picking program that identified the largest positive and negative peaks (relative to the 100 ms pre-stimulus baseline) within a pre-defined latency range at a pre-specified electrode site that were determined by visual inspection of grand mean waveforms. The pre-defined latency ranges and sites of maximum amplitude were defined as follows: N2 (300-500 ms, Cz) and P3 (290-580 ms, Pz). The latency of each component was locked to the latency at the pre-specified site of maximum amplitude.

To calculate pre-stimulus alpha, time domain data were divided into epochs defined as 500 ms pre-stimulus to stimulus onset (0 ms) and limited to correct responses only. Epochs were baseline corrected using the average amplitude across the entire epoch. An ocular artefact rejection procedure based on the vEOG channel (Semlitsch et al., 1986) was used to reduce the effect of eye movements, and an artefact rejection procedure removed trials with activity exceeding  $\pm 120$   $\mu$ V in the F3, F4 or Fz sites. The remaining data were low pass filtered (zero phase shift, 0-30 Hz, 48 dB/oct roll-off). A

fast Fourier transform (FFT) using a Hanning window of 10% length was computed across the 500 ms epochs to yield a power spectrum with a frequency resolution of 2 Hz. Due to resolution constraints the alpha range was defined as 8-14 Hz. Pre-stimulus alpha data were analysed at the Pz electrode site.

## 2.5 Statistical Analyses

An outlier analysis was used to identify values in the ERP data variables that were two times greater or smaller than the interquartile range, and these values were removed from the dataset. The remaining data were subject to Little's test which confirmed that the missing data were missing completely at random (MCAR),  $\chi^2(5927, N = 29) = 477.96, p = 1.000$ . No one variable had greater than 10 per cent of data points missing, therefore, all missing data were estimated using an Estimation-Maximisation (EM) imputation algorithm (Dempster et al., 1977).

Demographic and psychometric data were analysed using a univariate analysis of variance (ANOVA) with Age, Reading Age, Oppositional, Inattention, Hyperactivity, AD/HD Index, Anxious/Depressed, Withdrawn/Depressed, Somatic complaints, Social problems, Thought problems, Attention problems, Rule breaking, Aggressive behaviour, Internalise, Externalise and Total as the within subjects factors, and Group as the between-subjects factor.

Behavioural data (errors of commission, errors of omission, reaction time) were analysed using a mixed-design multivariate analysis of variance (MANOVA) with Group as the between subjects factor (control, AD/HD), and Event-rate (fast, medium, slow) and Stimulus (congruent, incongruent, neutral, target-alone) as the within-subjects factors. ERP component amplitudes were also analysed using a mixed-design MANOVA with Group as the between subjects factor, and Event-rate, Stimulus type and Sagittal (Fz, Cz, Pz) as the within-subjects factors. ERP component latencies were examined in the same manner as amplitudes but without the Sagittal factor. Planned contrasts within the Stimulus factor compared the neutral stimulus with the target-alone, incongruent and congruent stimuli individually, and within the Event-rate factor compared the medium event-rate with the fast and slow event-rates individually. Polynomial planned contrasts for the Sagittal factor compared Fz with Pz, and the mean of Fz and Pz with Cz, to assess the topographic distribution of each component.

In cases where Mauchly's test indicated that the assumption of sphericity was violated, degrees of freedom were adjusted using Greenhouse-Geisser estimates of sphericity and reported as such. Planned contrasts did not exceed the degrees of freedom for each effect, therefore no alpha adjustment for multiple comparisons was necessary. Additionally, planned comparisons were specified *a priori* and critical values were corrected using a Bonferroni correction, therefore, *post*

*hoc* alpha adjustments were not required (Tabachnick and Fidell, 1996). Values that are marginally significant ( $p < .10$ ) are reported in addition to those that are statistically significant ( $p < .05$ ).

### 3. Results

#### 3.1. Group Characteristics

Table 1 shows the means for the psychometric data for each group. Although the AD/HD group scored significantly lower than the control group in reading age and IQ, these were not included as covariates in order to preserve the clinical relevance of these data. The AD/HD group scored significantly higher on almost all subscales of the CPRS-R and CBCL, and in the clinical range for all subscales of the CPRS-R.

-- Insert Table 1 about here --

#### 3.2 Task Performance

Task performance data are shown in Figure 1. Across conditions, RT was shorter for the control than AD/HD group (675.37 vs. 745.33 ms), although this effect was only marginally significant,  $F(1,27) = 2.93$ ,  $p = .098$ ,  $\eta_p^2 = .098$ . RT was significantly longer to incongruent than neutral stimuli (834.64 vs. 677.79 ms),  $F(1,27) = 127.68$ ,  $p < .001$ ,  $\eta_p^2 = .825$ . However, no Stimulus x Group interaction was found,  $F(3,81) = .26$ ,  $p = .851$ ,  $\eta_p^2 = .010$ . Furthermore, RT was significantly shorter at the fast than medium event-rate (655.18 vs. 731.32 ms),  $F(1,27) = 12.12$ ,  $p = .002$ ,  $\eta_p^2 = .310$ . No difference was found between the medium and slow event-rate ( $M = 744.56$  ms),  $F(1,27) = .74$ ,  $p = .396$ ,  $\eta_p^2 = .027$ . No Event-Rate x Group interaction was found,  $F(2,54) = .381$ ,  $p = .685$ ,  $\eta_p^2 = .014$ .

-- Insert Figure 1 about here --

Across conditions, more omission errors were made by the AD/HD than control group (1.53 vs. 0.37), although this effect was only marginally significant,  $F(1,27) = 3.65$ ,  $p = .067$ ,  $\eta_p^2 = .119$ . Significantly more errors of omission occurred to incongruent compared to neutral stimuli (1.35 vs. 0.81),  $F(1,27) = 7.37$ ,  $p = .011$ ,  $\eta_p^2 = .215$ . Additionally, a Stimulus x Group interaction was found,  $F(1.86, 50.23) = 5.07$ ,  $p = .011$ ,  $\eta_p^2 = .158$ , indicating that the incongruent>neutral effect was significantly larger in the AD/HD than control group (*M difference* 1.00 vs. 0.09),  $F(1,27) = 5.16$ ,  $p = .031$ ,  $\eta_p^2 = .161$ . The Rate x Stimulus interaction,  $F(2.96, 80.02) = .703$ ,  $p = .551$ ,  $\eta_p^2 = .025$ , and Rate x Stimulus x Group interaction,  $F(6,162) = 2.03$ ,  $p = .117$ ,  $\eta_p^2 = .070$ , were not significant.

However, Bonferroni-adjusted pairwise comparisons revealed that at the fast event-rate, more omission errors were made to incongruent stimuli in the AD/HD group than control group (2.21 vs. 0.47),  $F(1,27) = 5.06, p = .033, \eta_p^2 = .158$ . Additionally, at the slow event-rate more omission errors were made to incongruent stimuli in the AD/HD than control group (2.57 vs. 0.53), although this difference was only marginally significant,  $F(1,27) = 3.58, p = .069, \eta_p^2 = .117$ .

The number of commission errors did not differ between the AD/HD ( $M = 1.74$ ) and control group (1.74 vs. 1.07),  $F(1,27) = 1.86, p = .184, \eta_p^2 = .064$ . Significantly more errors of commission occurred to incongruent than neutral stimuli (3.13 vs. 0.89),  $F(1,27) = 32.12, p < .001, \eta_p^2 = .543$ . Furthermore there were significantly more errors of commission in the fast than medium event-rate (1.85 vs. 1.16),  $F(1,27) = 10.10, p = .004, \eta_p^2 = .272$ , although no difference was found between the medium and slow event-rate ( $M = 1.21$ ),  $F(1,27) = .17, p = .683, \eta_p^2 = .006$ . The Event-Rate x Group interaction was marginally significant,  $F(1.51,40.48) = 3.10, p = .069, \eta_p^2 = .103$ . Planned contrasts revealed that the degree to which more commission errors were made at the medium event-rate than the slow event-rate was larger for the AD/HD than control group ( $M$  difference 0.23 vs. -0.33),  $F(1,27) = 5.31, p = .029, \eta_p^2 = .164$ .

### 3.3 Electrophysiological Measures

Grand mean ERPs for each Group at each event-rate and for each stimulus are shown in Figure 2 and 3 respectively. N2 peaked 393.86 ms after stimulus onset and was maximal in amplitude centrally (Cz>Fz/Pz),  $F(1,27) = 111.91, p < .001, \eta_p^2 = .806$ , across all conditions. N2 latency did not differ between stimuli,  $F(3,81) = 1.26, p = .295, \eta_p^2 = .044$ , or event-rates,  $F(2,54) = .094, p = .910, \eta_p^2 = .003$ .

-- Insert Figure 2 and 3 about here --

Overall, N2 amplitude did not differ between the AD/HD and control group (-4.52 vs. -3.58  $\mu V$ ),  $F(1, 27) = 2.60, p = .118, \eta_p^2 = .088$ . N2 amplitude was significantly larger to incongruent than neutral stimuli (-4.78 vs. -3.99  $\mu V$ ),  $F(1, 27) = 5.50, p = .027, \eta_p^2 = .169$ , an effect that did not differ between groups,  $F(3, 81) = .54, p = .659, \eta_p^2 = .019$ . No main effect of Event-rate,  $F(2, 54) = 1.02, p = .366, \eta_p^2 = .037$ , and no Event-rate x Group interaction,  $F(2, 54) = .96, p = .391, \eta_p^2 = .034$ , were found, however, in the slow event-rate N2 amplitude was significantly larger for the AD/HD than control group (-4.76 vs. -3.43  $\mu V$ ),  $F(1, 27) = 4.39, p = .046, \eta_p^2 = .140$ .

P3 peaked 489.53 ms after stimulus onset and was maximal in amplitude centro-parietally (Pz > Fz:  $F[1,27] = 46.41, p < .001, \eta_p^2 = .632$ ; Cz > Fz/Pz:  $F[1,27] = 21.66, p < .001, \eta_p^2 = .445$ ). P3 peaked later to incongruent than neutral stimuli (514.84 vs. 487.65 ms),  $F(1,27) = 8.26, p = .008, \eta_p^2 = .235$ .

= .234. However, no Stimulus x Group interaction was found,  $F(3,81) = 1.12, p = .345, \eta_p^2 = .040$ .

P3 amplitude was larger at the fast compared to medium event-rate (11.77 vs. 4.06  $\mu V$ ),  $F(1,27) = 49.74, p < .001, \eta_p^2 = .648$ . However, no difference was found between the medium and slow event-rate ( $M = 3.69 \mu V$ ),  $F(1,27) = .19, p = .668, \eta_p^2 = .007$ . No Rate x Group interaction was found,  $F(2,54) = 1.47, p = .238, \eta_p^2 = .052$ . The extent to which P3 amplitude was larger at Pz than Fz was larger at the fast than at medium event-rate,  $F(1,27) = 13.56, p = .001, \eta_p^2 = .331$ . Similarly, the extent to which P3 amplitude was larger at Pz than Fz was larger at the medium than slow event-rate,  $F(1,27) = 9.77, p = .004, \eta_p^2 = .266$ .

A main effect of Stimulus was found for P3 amplitude,  $F(3,81) = 3.45, p = .020, \eta_p^2 = .113$ . P3 amplitude was larger to incongruent than neutral stimuli (8.64 vs. 6.04  $\mu V$ ),  $F(1,27) = 5.80, p = .023, \eta_p^2 = .117$ . No Stimulus x Group interaction was found,  $F(3,81) = .33, p = .805, \eta_p^2 = .012$ .

Across conditions, pre-stimulus alpha power did not differ between the control and AD/HD group (5.41 vs. 5.60  $\mu V^2$ ),  $F(1,26) < .01, p = .953, \eta_p^2 = .006$ . Pre-stimulus alpha power was significantly greater at the fast than medium event-rate (5.98 vs. 5.33  $\mu V^2$ ),  $F(1,26) = 6.20, p = .019, \eta_p^2 = .187$ , however, no difference was found between the medium and the slow event-rate ( $M = 5.21 \mu V^2$ ),  $F(1,26) = .697, p = .411, \eta_p^2 = .026$ . Planned contrasts revealed that the difference in pre-stimulus alpha power between the medium and slow event-rates was larger for the AD/HD than control group (*M difference* 0.382 vs. -0.14  $\mu V^2$ ), although the difference was only marginally significant,  $F(1,27) = 3.25, p = .082, \eta_p^2 = .108$ .

## 4. Discussion

The aim of the present study was to examine the ability of children with AD/HD to modulate their energetic state during an interference control task. To this end, a flanker task was employed with three event-rates, and task performance and ERPs were analysed as indices of state-regulation ability. Analysis of demographic data confirmed that the control and AD/HD groups were uniquely defined and successfully matched for age. The AD/HD group scored significantly lower than the control group in Reading Age and IQ, which are common group differences (Barkley, 1997; Faraone et al., 1993).<sup>1</sup>

### 4.1 Task Performance

In line with previous studies (e.g. Heil et al., 2000; Johnstone et al., 2009; Mullane et al.,

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<sup>1</sup> These group differences are likely due to the inability of children with AD/HD to apply their knowledge and abilities to the psychometric instruments, as opposed to any inherent differences. Therefore, reading age and IQ were not included as covariates in order to preserve the clinical relevance of these data.



2009), conflicting flankers had an effect on commission error rate, with significantly more commission errors occurring to incongruent stimuli than neutral stimuli. The number of commission errors made did not differ between groups. Moreover, contrary to studies that found a larger interference (incongruent>neutral) effect for children with AD/HD (e.g. van Meel et al., 2007), the present study found no difference between the control and AD/HD groups.

Alternatively, results indicated that the AD/HD group tended to make more errors of omission than the control group. Furthermore, more errors of omission were made to incongruent stimuli compared to neutral stimuli, an effect that was larger for the AD/HD than control group. Together, these results suggest that children with AD/HD are able to focus attention on the target stimulus and suppress conflicting flankers as effectively as typically-developing children. However, children with AD/HD are unable to constantly attend to the task at hand, particularly to difficult incongruent stimuli.

Event-rate had no overall effect on commission errors, adding to mounting evidence that commission errors do not follow the expected U-shaped trend (e.g. Börger and van der Meere, 2000; Scheres et al., 2001). However, it was found that the control group made more errors at the slow than medium event-rate, whereas the AD/HD group made more errors at the medium than slow event-rate. This result is surprising considering performance deficits in AD/HD most commonly occur to slow event-rates (Sonuga-Barke et al., 2010; Wiersema et al., 2006b). However, as energetic state is both idiosyncratic and task-dependent, the findings of the present study may be attributable to the unique task requirements of the flanker task (Sonuga-Barke et al., 2010). Conversely, as mentioned previously, a larger interference effect (incongruent>neutral) was found for omission errors in the AD/HD compared to the control group. This was due to the fact that, compared to the control group, the AD/HD group tended to make more errors of omission to incongruent stimuli at both the fast and slow event-rates but not the medium event-rate. This is consistent with the CEM and suggests that AD/HD children fail to constantly attend to the task in situations where they are required to modulate their energetic state.

## **4.2 ERPs**

In the present study, the N2 component was enhanced to incongruent compared to neutral stimuli, in line with both ERP studies (Heil et al., 2000; Johnstone et al., 2009; Johnstone et al., 2010) and computational models (Yeung et al., 2004; Yeung and Cohen, 2006) of the flanker N2. This enhancement of N2 has been explained in terms of conflict monitoring (Yeung et al., 2004), with N2 amplitude indexing a conflict monitoring process that occurs due to the coactivation of incompatible responses in the incongruent condition (i.e., simultaneous left and right responses;

Kopp et al., 1996). Following this, it was expected that a conflict-processing deficit in children with AD/HD would manifest in the amplitude difference between incongruent and neutral stimuli (i.e., the effect of conflicting response information compared to typical non-conflict response processing). Although it seems counterintuitive, correct responses are associated with high levels of conflict processing. More processing serves to resolve conflicting response information in order to respond correctly to the target stimulus (Yeung and Nieuwenhuis, 2009).

Therefore it was predicted that children with AD/HD would present with a smaller incongruent>neutral N2 amplitude difference than typically-developing children. However, results indicated that this effect did not differ between the groups. It seems, therefore, that both groups were successfully able to focus their attention on the target stimulus and inhibit the effect of flankers. For example, Swick and Turken (2002) found that a patient with an anterior cingulate cortex (ACC) lesion lacked the ability to selectively focus on the target stimulus. This resulted in more commission errors and was concomitant with enhanced N2 amplitude to incongruent stimuli. Therefore, the failure to find incongruent>neutral N2 differences fits well with behavioural results in the present study indicating that both groups made an equivalent number of commission errors to incongruent stimuli.

As the present study kept the response window constant, it was predicted that there would be no event-rate differences in N2 amplitude or latency. Generally the results supported this hypothesis. However, the AD/HD group did present with appreciably larger N2 amplitude at the slow event-rate. This effect was due to an enhanced N2 amplitude to target-alone stimuli that was absent in the control group. The conflict N2 is insensitive to target-alone stimuli (Kopp et al., 1996) and thus should not be associated with enhanced target-alone N2 amplitudes. Alternatively, this effect can be attributed to contamination by the 'novelty N2', a component which is sensitive to perceptually deviant and anomalous stimuli. In the flanker task the novelty N2 is typically enhanced to neutral stimuli (Folstein and Van Petten, 2008). However, in the present study the participants may have perceived 5-item arrays (i.e., neutral, congruent and incongruent stimuli) as prevailing, and 1-item stimuli (i.e., target-alone stimuli) as deviant. Additionally, the novelty N2 has a similar timing and topography to the conflict N2 in the flanker task involving children with AD/HD (Johnstone et al., 2010), making it a possible explanation for these results.

In line with expectations, the P3 peaked later to incongruent stimuli than neutral stimuli, suggesting that incongruent stimuli required a longer time to evaluate than other stimuli (Coles et al., 1985; Gehring et al., 1992; Johnstone et al., 2010). Furthermore, as predicted, P3 amplitude was larger to incongruent stimuli than to neutral stimuli. Generally, larger P3 amplitudes to incongruent stimuli in the flanker task are attributed to effort allocation (Banaschewski et al., 2005; Johnstone et

al., 2010) and more effort is required in order to respond appropriately to incongruent stimuli than other stimuli. This theory is consistent with evidence of an enhanced P3 to Nogo stimuli in the Go/Nogo task (Bokura et al., 2001) and to stop signals in the Stop-Signal task (Kok et al., 2004). Therefore, in the flanker task it is likely that the enhanced P3 reflects effortful processing required by the high interference control demands of incongruent stimuli. The present study failed to find any group differences for incongruent stimuli suggesting that children with AD/HD and typically-developing children are able to apply effort equally effectively when they each respond *correctly*.

As predicted the present study also found that P3 amplitudes were larger and more parietal at the fast than medium and slow event-rates. All three event-rates included a 1500 ms response window. In line with the effort allocation interpretation of the flanker P3, keeping the response window constant ensured that event-rate manipulations altered general energetic demands of the task, whilst keeping interference control demands constant. It is probable that the fast event-rate required a greater amount of effort than the medium and slow event-rates, and thus only the fast event-rate was accompanied by larger P3 amplitudes.

It was also predicted that the differences in P3 amplitude between the medium and fast event-rates would be smaller in the AD/HD than control group. However, in contrast to other studies, the present study failed to find any group differences in P3 amplitude. For example, using a three event-rate Go/Nogo task, Benikos and Johnstone (2009) found that P3 amplitude was larger in the fast than slow event-rate for the control group, but found no difference in the AD/HD group. In terms of the effort allocation theory of the P3, those results suggest that only the control group was able to successfully allocate more effort to the highly demanding fast event-rate condition. Following this, it would be tempting to suggest that the results of the present study indicate that both groups were able to allocate additional effort successfully for fast event-rate. However, erroneous responses do not contribute to the group P3 averages, thus group differences cannot be inferred from P3 amplitudes. Indeed, compared to the control group, the AD/HD group made significantly more omission errors to incongruent stimuli at the fast and slow event-rates. Therefore, it is possible that they were unable to allocate effort appropriately at those event-rates. Analyses of P3 amplitudes for both correct and incorrect responses would reveal more about group differences in effort allocation.

In contrast to expectations, it was found that pre-stimulus alpha power was larger in the fast than medium event-rate, although the medium and slow event-rates did not differ. According to the theory that pre-stimulus alpha is an inverse measure of cortical arousal (e.g. Carp and Compton, 2009), these results suggest that the fast-event rate was associated with lower arousal than the medium and slow event-rates. This result is counterintuitive, and not in line with evidence that fast event-rates induce a highly aroused state (Hockey et al., 1986). However, evidence has recently

emerged indicating that high levels of alpha power may reflect a state of active cortical inhibition. According to the inhibition-timing hypothesis, activity in the alpha band is caused by synchronisation of rhythmic oscillations of inhibitory cells (Klimesch et al., 2007). Using two different inhibition tasks, Min and Herrmann (2007) determined that pre-stimulus alpha power was associated with task difficulty. They found that a higher pre-stimulus alpha power indicated that the task required a greater amount of inhibition. It was concluded that pre-stimulus alpha power reflected top-down processing preparing for upcoming inhibition. Therefore, it may be the case here that the fast event-rate increased task requirements, and was associated with a higher pre-stimulus alpha power. Following this line of reasoning, it is likely that the slow event-rate unsuccessfully altered the participants' energetic state as the present study failed to find differences in alpha power between the medium and slow event-rates. Indeed, this assertion is supported by the fact that no significant differences in task performance were found between the medium and slow event-rates.

Finally, it was also predicted that, compared to controls, children with AD/HD would be over-aroused at the fast event-rate and under-aroused at the slow event-rate. As this hypothesis was based upon the premise that alpha power was an inverse measure of cortical arousal, it is not surprising that it was not supported by the results of the present study. No group differences were found for pre-stimulus alpha power at each event-rate. However, whilst the control group presented with higher alpha power at the medium event-rate than at the slow event-rate, the control group showed the opposite effect. It is notable that the patterns of pre-stimulus alpha correspond precisely with the pattern of commission errors made by the groups. If commission errors are an indication of task difficulty, then it is appropriate that pre-stimulus alpha power was higher when the task is more difficult (i.e., at the fast event-rate).

Overall, the findings of the present study indicate that the inability of children with AD/HD to modulate their energetic state results in a failure to attend to the task for prolonged periods of time (indicated by a greater number of omission errors) but not an incapacity to selectively attend to target stimuli when performing the task (indicated by an equal number of commission errors). Moreover, the inadequacy of the energetic state manipulation notwithstanding, it was found that the AD/HD group followed the predicted U-shaped pattern of task performance for omission errors only. This has two implications: Firstly, it undermines the accuracy of fixed deficit models of AD/HD. Fixed-deficit models claim that underperformance in children with AD/HD occurs independently of event-rate, yet the present results clearly demonstrate the influence of event-rate on error rates. Secondly, it adds to the CEM literature insofar as only omission errors follow the pattern predicted by the CEM. This suggests that the energetic state modulation dysfunction in children with AD/HD results in hypovigilance (i.e., an inability to sustain attention) as opposed to poor task performance *per se*.

These results provide have practical implications for children with AD/HD who often show an inability to sustain attention and inhibit distractions in classroom situations. It is conjectured that children with AD/HD would benefit from a moderate rate of presentation of classroom material, the minimization of distractions and cues aimed at keeping their attention on the task at hand. In particular, due to the fact that children with AD/HD were found to perform the task equally as well as typically-developing children in the present study, they may benefit from reinforcement upon task completion, thereby promoting task vigilance.

## **Acknowledgements**

We thank the children who participated and their parents for their time and patience, Aneta Dimoska and Val Markovska for assistance with data collection, and the anonymous reviewers for their helpful comments on this manuscript. This study was funded by an Australian Research Council Discovery Project Grant (DP0559048).

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**Table 1***Mean scores for psychometric variables, CPRS-R and CBCL.*

Measure	Controls	AD/HD	F
Age	11.66 (2.11)	11.09 (2.15)	.51
Reading Age	10.96 (2.33)	9.04 (1.64)	5.73*
IQ	115.60 (11.65)	99.54 (17.99)	8.08**
<i>CPRS-R</i>			
Oppositional	47.33 (5.78)	<b>66.93</b> (10.37)	40.27***
Inattention	49.13 (5.88)	<b>73.43</b> (7.65)	92.68***
Hyperactivity	47.13 (3.78)	<b>70.67</b> (10.83)	61.93***
AD/HD Index	46.40 (5.14)	<b>72.50</b> (8.40)	103.48***
<i>CBCL</i>			
Anxious/Depressed	54.27 (6.57)	60.14 (8.63)	4.39*
Withdrawn/Depressed	53.80 (5.33)	57.21 (6.64)	2.35 <sup>†</sup>
Somatic complaints	53.27 (3.79)	58.93 (7.98)	6.10*
Social problems	52.87 (3.83)	66.50 (8.16)	33.92***
Thought problems	52.33 (3.02)	63.79 (10.66)	15.97***
Attention problems	51.80 (2.14)	69.57 (6.90)	90.30***
Rule breaking	52.13 (4.22)	58.36 (6.42)	9.65**
Aggressive behaviour	53.00 (6.46)	63.00 (6.68)	16.80***
Internalise	49.53 (9.27)	58.93 (10.96)	6.24*
Externalise	47.27 (8.94)	61.79 (6.83)	23.89***
Total	45.93 (9.64)	65.00 (8.49)	31.75***

*Note:* Standard deviations are provided in parentheses. CPRS-R: a score of 60-64 is considered borderline and a score of greater than 65 is considered clinical (bolded). CBCL: a score of 67-69 is considered borderline and a score greater than 70 is considered clinical (bolded).

<sup>†</sup>  $p < .10$ , \*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$ .

Figure 1

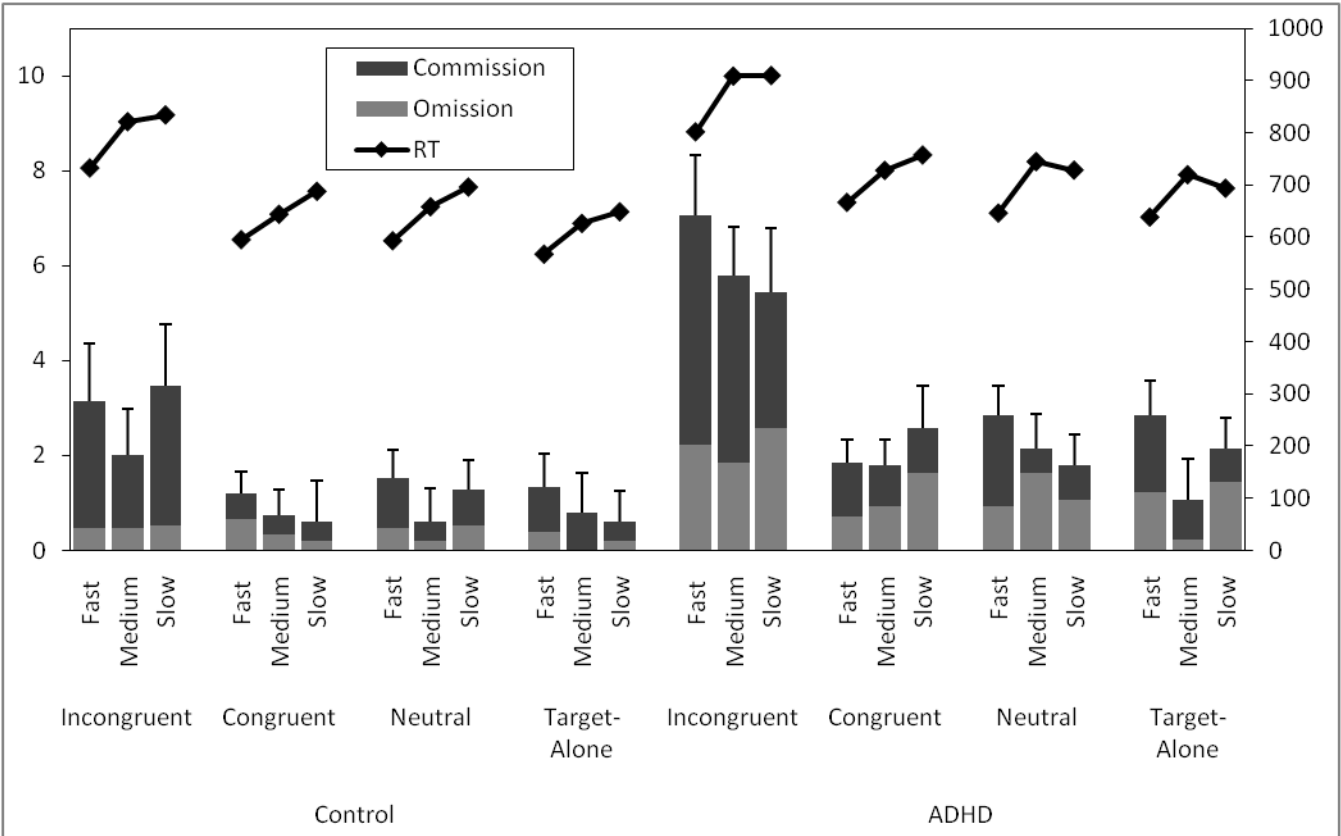


Figure 1. Means for errors and RT to each stimulus at each event-rate for the control group (left) and ADHD group (right). Vertical lines show +1 standard error of the mean for total errors. Right Y-axis scale is ms, left Y-axis scale is number.

**Figure 2**

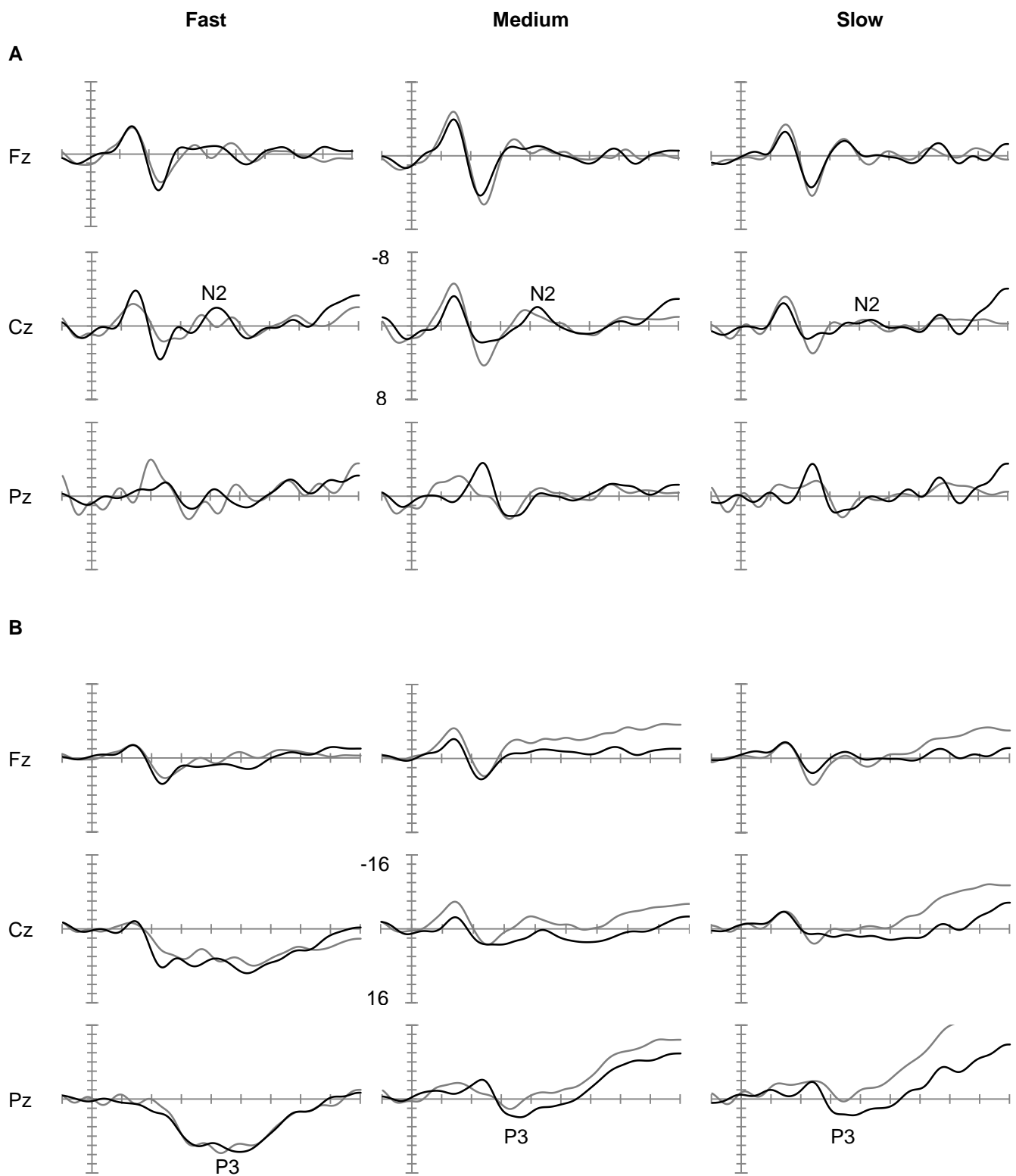


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**Figure 3**

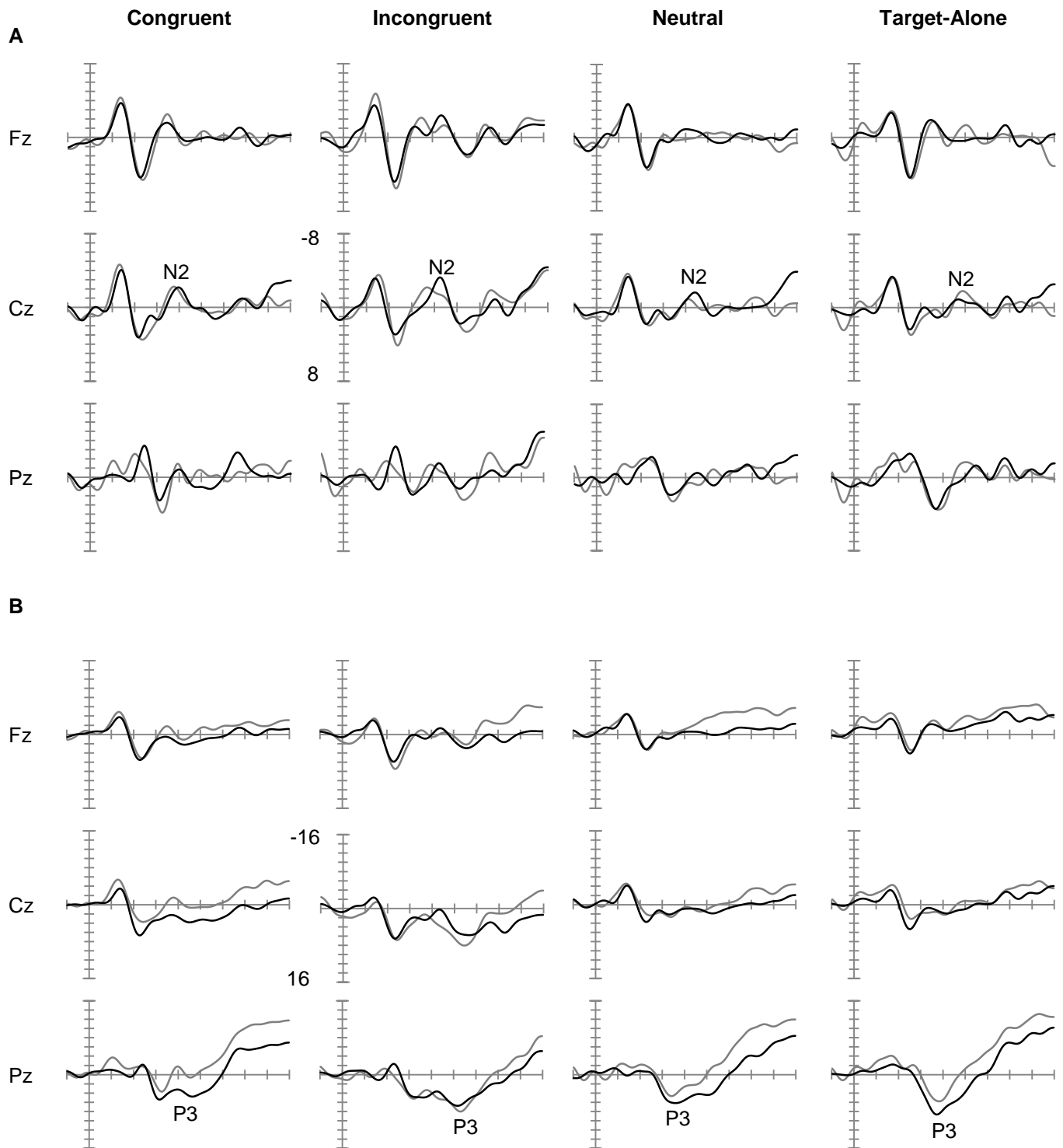


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